A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement

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*The names of the task force, which consisted of 49 people from 7 countries, are given on the *BMJ*'s website

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Chairman's foreword

In 1997 the research committee of the Perinatal Society of Australia and New Zealand competitively funded a special initiative to bring together the modern literature on the causation of cerebral palsy, and to try to define an objective template of evidence to better identify cases of cerebral palsy where the neuropathology began or became established around labour and birth. Recently there have been many advances in a wide variety of scientific areas associated with cerebral palsy, and thus this multidisciplinary review may benefit research into the causation and prevention of cerebral palsy and may also help those who offer expert opinion when counselling in this area or giving such opinion in court.

The corresponding task force was open to anyone who could make a scientific contribution to understanding in this area. The task force had representation from a wide range of clinical and scientific specialties. Submissions were sought from the society's 1000 members, which include scientists, pathologists, obstetricians, neonatalogists, midwives, neonatal nurses, and epidemiologists. International contributions were sought from those identified from the current literature as contributing to this area through peer reviewed research. They were not preselected for their views, and they were invited to join the corresponding task force. Some international members joined later in the discussion process as word of this open debate reached them.

During 1997 and 1998 multiple online electronic conferences were held, and in March 1998 many of the task force members were able to participate in a workshop in Alice Springs, Australia to discuss the fourth draft of the statement. Drafts of the statement were circulated and debated, with the sixth draft being discussed at an international telephone conference in October 1998. The paper continued to be redrafted eight times until consensus was reached. No opinion was excluded from the debate, but the statement only includes discussion that has a reasonable scientific basis and can be referenced. All members agree that updated reports will be required within a few years when further information is published. It is hoped that other international researchers in this area will offer to assist in future statements. The final draft of the statement was sent to the professional colleges and scientific societies to which the task force members belong. To date no such group has declined to support the statement or has disputed its final content. Box 1 lists those professional bodies endorsing the statement at the time of publication.

Box 1—Supporters of the consensus statement

American College of Obstetricians and Gynecologists
American Gynecological and Obstetrical Society
Australian College of Midwives
Hong Kong Society of Neonatal Medicine
Institute of Obstetrics and Gynaecology of the Royal College of Physicians of Ireland
International Society of Perinatal Obstetricians
New Zealand College of Midwives
Paediatric Society of New Zealand
Perinatal Society of Australia and New Zealand
Royal Australasian College of Physicians, Paediatric Division
Royal Australian College of General Practitioners
Royal Australian College of Obstetricians and Gynaecologists
Royal College of Obstetricians and Gynaecologists
Royal College of Pathologists of Australasia
Royal New Zealand College of Obstetricians and Gynaecologists
Society of Obstetricians and Gynaecologists of Canada

Consensus was reached with some difficulty in two areas. Firstly, the current validity of neuroimaging in the infant to retrospectively determine the precise perinatal timing, pathology, or cause of the abnormalities seen on imaging. The task force awaits the publication of strong data, using the criteria suggested in its template, to define an acute intrapartum hypoxic event to validate specific neuroimaging appearances that occur as a result of acute intrapartum hypoxia. These images must be different from those that arise after chronic asphyxial or non-asphyxial causes of cerebral palsy in order to be of help in determining intrapartum timing.

The second area of debate was terminology, and in particular the term "non-reassuring fetal status" was debated. It has been adopted in the consensus statement rather than the term "fetal distress" as clinical signs often poorly predict a compromised fetus, and continued use of this latter term may

encourage wrong assumptions or inappropriate management.

The task force acknowledges that some cases of cerebral palsy probably originate in labour. By defining these cases it should also help to focus research on the many antenatal causes of cerebral palsy and their prevention as well as the prevention of intrapartum asphyxia, which has been the major clinical goal to date.

Introduction

New insights into the origins of cerebral palsy have recently transformed the old concept that most cases of cerebral palsy begin in labour. There are many causes including developmental abnormalities,[w1 w2] metabolic abnormalities,[w3] autoimmune and coagulation disorders,[w4] and infections,[w5] in addition to trauma and hypoxia (asphyxia)[w6 w7] in the fetus and newborn. Efforts to identify possible causes of cerebral palsy require skills from many disciplines.

Contrary to previous beliefs and assumptions, clinical epidemiological studies indicate that in most cases the events leading to cerebral palsy occur in the fetus before the onset of labour, or in the newborn after delivery.[w8 w9] In a given clinical scenario, however, it is difficult to assess which features indicate causes contributing to the adverse outcome. This is particularly true before delivery when the clinical measures used for the assessment of fetal wellbeing are almost always indirect and generally inadequate to assess fetal brain function.

The problem for the individual case is that it is very difficult to identify retrospectively antenatal causes of cerebral palsy. Conversely, damaging hypoxia (asphyxia) during labour can be suspected from many clinical signs, none of which are specific to damaging hypoxia and which could, therefore, reflect other conditions in the fetus.[w10] By combining some of these clinical signs with certain objective investigations, hypoxia during labour may be more reliably ascertained. The timing of the onset of antenatal or intrapartum hypoxia requires further evidence that is discussed in this statement.

Terminology

This task force reiterates a previous consensus statement[w11] that the terms fetal distress and birth asphyxia are inappropriate and should not be used clinically. This opinion is endorsed by the American College of Obstetricians and Gynecologists[w12] and the Society of Obstetricians and Gynaecologists of Canada.[w13] The term fetal distress should be replaced by the term non-reassuring fetal status and a description of the clinical sign or test that led to that conclusion—for example, pathological fetal acidaemia as defined by an umbilical arterial cord pH of less than 7.0.

The term asphyxia is defined experimentally as impaired respiratory gas exchange accompanied by the development of metabolic acidosis. It is usually reserved for experimental situations in which these changes can be accurately established. In the clinical context fetal asphyxia is progressive hypoxaemia and hypercapnia with a significant metabolic acidaemia.[w14 w15] In practice, the timing of the onset and the progression of these changes can be difficult or impossible to ascertain. When possible, the timing of factors should be described as fetal or antenatal if they occur before the onset of labour; as intrapartum if they occur or recur between onset of labour and complete expulsion of the baby, and neonatal if they occur after the birth has been completed. Perinatal asphyxia can be used when the timing is uncertain. Factors may also be described as acute (occurring within a brief time period such as hours) or chronic (commencing in one period and continuing into subsequent periods, over days or weeks) and as continuous or intermittent.

Neonatal encephalopathy is a clinically defined syndrome of disturbed neurological function in the infant at or near term during the first week after birth, manifested by difficulty with initiating and maintaining respiration, depression of tone and reflexes, altered level of consciousness, and often seizures.[w16] It is less clear which clinical signs indicate disturbed neurological function in very preterm infants. We have chosen not to use the diagnosis hypoxic ischaemic encephalopathy in this template as hypoxia and ischaemia have often not been proved and have been assumed from a variety of clinical markers that do not accurately reflect hypoxia and ischaemia of either acute or chronic origin.[w16] Over 75% of cases of neonatal encephalopathy have no clinical signs of intrapartum hypoxia.[w17-w20]

Problems in defining the cause and timing of the neuropathology causing cerebral palsy

Cerebral palsy, which is characterised by non-progressive, abnormal control of movement or posture, is not diagnosed until months or years after birth. Retrospective review of the pregnancy records often cannot show any obvious antenatal cause because fetal brain development and function cannot currently be routinely visualised or monitored. Complications occurring in the antepartum period are common and important causes of cerebral palsy. Epidemiological studies suggest that in about 90% of cases intrapartum hypoxia could not be the cause of cerebral palsy and that in the remaining 10% intrapartum signs compatible with damaging hypoxia may have had antenatal or intrapartum origins.[w21 w22] These studies show that a large proportion of cases are associated with maternal and antenatal factors such as prematurity, intrauterine growth restriction, intrauterine infection, fetal coagulation disorders, multiple pregnancy, antepartum haemorrhage, breech presentation, and chromosomal or congenital anomalies.[w1-w11 w23-w25]

Signs of fetal compromise such as changes in fetal heart rate and passage of meconium are neither sensitive nor specific to any particular cause and only sometimes indicate damaging intrapartum hypoxia.[w26] When metabolic acidaemia has been conclusively proved by fetal blood gases and umbilical arterial cord or very early neonatal blood gases, or both, it remains to be proved whether this is attributable to a chronic or intermittent hypoxia of longstanding duration—for example, days and weeks—or whether a de novo acute hypoxia has occurred during labour or birth in a previously healthy fetus. Intrauterine growth restriction

may sometimes be associated with chronic hypoxia and cerebral palsy.[w27] Animal studies show that induced, prolonged placental insufficiency in the last third of gestation resulting in persistent moderate fetal hypoxaemia disrupts myelination and the growth of the cerebellum.[w28] They also show that, in mid-gestation, an episode of 12 hours' hypoxaemia is enough to cause damage to white matter and neuronal death in the hippocampus, cerebral cortex, and cerebellum.[w29]

Intrapartum complications play an infrequent role in the causation of cerebral palsy. If a fetus has experienced neurological damage or maldevelopment during pregnancy, the neurological lesions, which are often multifocal, may affect parts of the fetal brain responsible for the autonomic nervous system that control such activities as heart rate and respiration. Reduced variability of the fetal heart rate, meconium staining seen at membrane rupture, low Apgar scores, and neonatal encephalopathy may all represent the first recognised signs of chronic neurological compromise. In a chronically compromised case, the intrapartum signs may precipitate an obstetric intervention such as an instrumental or caesarean delivery in the hope that the pathology is of recent onset and still reversible. Retrospectively the presence of these signs and the decision of the carers to act to prevent possible acute compromise may mistakenly be taken as evidence of acute compromise.

It is not currently possible to recognise the point at which cerebral damage becomes irreversible in the case of an intermittent type of fetal asphyxia or intrauterine growth restriction. It is possible that the point of irreversible neurological injury could be reached in labour if the fetus has been able to compensate adequately until that time. Intrauterine growth restriction of potential pathological significance can be difficult to detect clinically, and randomised trials are awaited to see if earlier delivery, when moderate to severe intrauterine growth restriction is suspected, reduces the incidence of cerebral palsy without a greater increase in the complications of prematurity.[w30 w31]

Criteria suggesting that acute intrapartum hypoxia was the cause of cerebral palsy

Box 2 shows the template of evidence required to suggest the occurrence of damaging intrapartum hypoxia sufficient to cause permanent neurological impairment. Taken together, criteria 4 to 8 help to time the hypoxic event to the intrapartum period.

Unavailable or contrary evidence

All three of the essential criteria are necessary before an intrapartum hypoxic cause of cerebral palsy can begin to be considered. If any one of the essential criteria is not met, it strongly suggests that intrapartum hypoxia was not the cause of the cerebral palsy. If blood gas data are not available, it cannot be assumed from other signs that hypoxia was present at birth since these signs lack specificity either individually or as a group.[w32-w34] When all three essential criteria are met it is then necessary to determine whether the hypoxia was acute or chronic. If evidence for some of criteria 4 to 8 is missing or contradictory, the timing of the onset of the neuropathology becomes increasingly in doubt. Individually, these latter criteria are only weakly associated with an acute intrapartum damaging hypoxic event because, with the exception of criterion 4 (a sentinel hypoxic event), they may be caused by other factors such as infection.[w5] Logically, most of the final five criteria would have to be present for the balance of probabilities to suggest an acute timing to the hypoxic event. Contrary evidence, rather than missing evidence—for example, a normal Apgar score at 5 minutes—would weigh against a serious acute event.

Box 2—Criteria to define an acute intrapartum hypoxic event

Essential criteria
1 Evidence of a metabolic acidosis in intrapartum fetal, umbilical arterial cord, or very early neonatal blood samples (pH < 7.00 and base deficit ≥ 12 mmol/l)
2 Early onset of severe or moderate neonatal encephalopathy in infants of ≥ 34 weeks' gestation
3 Cerebral palsy of the spastic quadriplegic or dyskinetic type

Criteria that together suggest an intrapartum timing but by themselves are non-specific
4 A sentinel (signal) hypoxic event occurring immediately before or during labour
5 A sudden, rapid, and sustained deterioration of the fetal heart rate pattern usually after the hypoxic sentinel event where the pattern was previously normal
6 Apgar scores of 0-6 for longer than 5 minutes
7 Early evidence of multisystem involvement
8 Early imaging evidence of acute cerebral abnormality

Comments on proposed criteria

Metabolic acidaemia

Although its presence does not define the timing of its onset, metabolic acidaemia at birth will have to be present if a potentially damaging intrapartum hypoxic event is postulated. Metabolic acidaemia at birth is, however, comparatively common (2% of all births), and the vast majority of such infants do not develop cerebral palsy.[w35]

A realistic cut off point for defining pathological fetal acidaemia that correlates with an increasing risk of neurological deficit has been found to be a pH of less than 7.00 and a base deficit of more than 16 mmol/l.[w36 w37] These are also the criteria agreed by the Society of Obstetricians and Gynaecologists of Canada[w13] and the American College of Obstetricians and Gynecologists.[w12] It is unlikely that acute acidosis of lesser severity under these levels could be directly associated with cerebral palsy. Occasional cases have, however, been reported where the base deficit has been in the range of 12-15, and thus an arterial base deficit of less than 12 mmol/l is a reasonable exclusion criterion.[w38] If evidence from fetal, umbilical arterial cord, or very early (less than 1 hour) neonatal blood gas does not exist, it is not possible to say that hypoxia or asphyxia caused or contributed to the other clinical signs. If records of both arterial and venous umbilical cord gases exist then a difference in partial pressure of

carbon dioxide of more than 25 mm Hg suggests an acute rather than chronic acidosis.[w39] However, the technique for measuring arterial versus venous gases is critical.[w40] Individual partial pressures of oxygen are not helpful in this context as they correlate poorly with fetal acidosis.[w39] A metabolic acidosis measured in the neonate can also reflect neonatal exposure to asphyxia attributable to difficult resuscitation, and thus neonatal gas results carry less weight in determining an intrapartum cause.

Neonatal encephalopathy

If an intrapartum insult has caused permanent brain damage in an infant of more than 34 weeks' gestation there will be abnormalities of behaviour in the neonatal period, usually of at least moderate severity and noted within 24 hours of delivery. However, moderate to severe encephalopathy after a non-reassuring intrapartum cardiotocograph is very uncommon, occurring in around 7 per 1000 such births—just twice the rate in the background population.[w41] Conversely, many cases of severe neonatal encephalopathy are not associated with intrapartum hypoxaemia.[w17-w20] Cerebral palsy associated with intrapartum events in infants born beyond 34 weeks' gestation is only rarely an outcome associated with milder grades of encephalopathy.[w42-w44] Infants with severe encephalopathy frequently have an adverse outcome.[w42] The outcome of those with moderate encephalopathy is less certain. The Sarnat grades[w45] of encephalopathy are commonly used, but there are some differences in opinion about the criteria defining moderate and severe encephalopathy.[w16] There is difficulty, short of the presence of seizure activity, in defining neonatal encephalopathy in infants of less than 34 weeks' at birth, where many of the diagnostic criteria described in infants near term would be part of the normal preterm course before 34 weeks (for example, difficulty initiating and maintaining respiration, abnormal tone, and feeding difficulties). Because of this and the absence of clear evidence in the literature, we can be less definite about the link between abnormal newborn behavioural states in the preterm infant and cerebral palsy attributable to an intrapartum cause.

Cerebral palsy type

Spastic quadriplegia and, less commonly, dyskinetic cerebral palsy are the only subtypes of cerebral palsy associated with acute hypoxic intrapartum events.[w42 w46] Spastic quadriplegia is not specific to intrapartum hypoxia. Only 24% of a population based series of children with moderate or severe spastic quadriplegia were thought possibly or very likely to have been affected by intrapartum events.[w46] Hemiplegic cerebral palsy, spastic diplegia, and ataxia have not been associated with acute intrapartum hypoxia. This is true also for intellectual disability, autism, and learning disorder in a child without spasticity. Any progression of neurological disability is not cerebral palsy by definition and is most unlikely to be secondary to birth events. Many conditions likely to be confused with cerebral palsy, for example, Rett syndrome and Angelman syndrome, should be excluded.[w47]

Sentinel hypoxic event

The healthy fetus has many special physiological mechanisms to protect it from recurrent transient mild hypoxic episodes that can occur during labour.[w48] For a neurologically intact fetus, uncompromised by chronic hypoxia, to sustain a neurologically damaging acute hypoxia a serious pathological sentinel hypoxic event has to occur. Examples are ruptured uterus, placental abruption, cord prolapse, amniotic fluid embolism, and fetal exsanguination from vasa previa or fetal-maternal haemorrhage. An antepartum or intrapartum hypoxic event can be silent. It is only when it is apparent or detectable that it helps to define the probable timing of the event and the determination of whether its sequelae might have been preventable.

Fetal heart rate

What little randomised controlled evidence is available suggests that use of electronic fetal monitoring does not prevent cerebral palsy.[w49] Even the observations most associated with cerebral palsy on electronic fetal monitoring—that is, multiple late decelerations and decreased beat to beat variability—should not be used to predict cerebral palsy as they have a false positive rate of 99.8%.[w50] Pre-existing neurological defects may cause a reduction or absence of fetal heart variability. The high frequency (up to 79%) of non-reassuring patterns on electronic monitoring of normal pregnancies in labour with normal fetal outcomes makes both the decision on the optimal management of the labour and the prediction of current or future neurological status of the fetus very difficult.[w51] Retrospective analysis of fetal heart rate traces where the neonatal outcome is known by the reviewer profoundly biases obstetricians who are asked to judge the appropriateness of care.[w52] Fetal heart rate patterns of potential severe fetal compromise merit early delivery where this can be achieved without undue risk to the health and life of the mother.

This task force endorses the statement by the US National Institute of Child Health and Human Development on electronic fetal monitoring,[w53] which presented recommendations for standardised definitions of each of the characteristics of the fetal heart rate pattern. The North American committee had a great deal of difficulty in reaching consensus on appropriate management of certain heart rate patterns, except for two. The first is that in the case of a fetus with a baseline heart rate within the normal range (110-160 beats/min) and moderate fetal heart rate variability (6-25 beats/min) and absent decelerations, the fetus is not at risk of acidaemia. The committee also stated that at the other extreme of fetal heart rate patterns, a fetus with absent fetal heart rate variability in the presence of persistent late or variable decelerations, or a bradycardia, has evidence of potentially damaging acidaemia. The committee further decided that, because of insufficient evidence, it was impossible at this stage to reach consensus on the management of all other patterns that are variants of the normal pattern. Such recommendations will have to await further research on the reliability, validity, and ability of monitoring as a means of avoiding adverse outcomes by prompting obstetric action.

Apgar scores

Apgar scoring is a quick and somewhat subjective method of assessing the condition of newborn infants. Low Apgar scores do not indicate the cause of the poor condition, which may result from many different factors of which acute intrapartum hypoxia is only one. By themselves, Apgar scores are poor predictors of neurological outcome. In particular, in preterm infants the Apgar score is highly limited in this respect.[w54] Although the risk of poor outcome increases with decreasing Apgar score and increasing duration of low scores, a 5 minute Apgar score below 4 but improving thereafter was associated with an increase in the risk of cerebral palsy from 0.3% to only 1% for births (birth weights of more than 2501 g) in the 1950s and 1960s.[w55] Currently, duration of low Apgar scores is more likely to indicate the effectiveness of resuscitation than to predict outcome.[w56]

Multisystem involvement

Multisystem involvement may include acute bowel necrosis, renal failure, hepatic injury, cardiac damage, respiratory complications, or haematological insult.[w57-w59] This requires testing over the early neonatal period (within 24 hours). Acute hypoxia usually affects all the vital organs and not just the brain but may occasionally occur without major dysfunction of other organs.[w60]

Neonatal brain imaging

Early cerebral oedema with or without intracerebral haemorrhage suggests recent morbidity. After an acute cerebral insult, oedema appears within 6-12 hours and clears by 4 days after the insult.[w61] Macroscopic abnormalities suggesting longstanding neurological changes may be visualised, but dysfunction that is at an intracellular level will be missed. Magnetic resonance imaging is currently the most informative imaging modality but requires the greatest resources and is not widely available in many countries.[w62] Although it may usefully predict future neurological disability in the neonate or infant, magnetic resonance imaging has not yet been validated (against well defined and well timed acute fetal asphyxial events) as a retrospective tool reliably defining the timing of the initial or main neuropathological event. Evidence from neonatal ultrasound is more often available, but false positive and false negative results are possible.[w63] Cerebral haemorrhage and oedema can also occur later in the neonatal period, in which case it is not the result of acute intrapartum hypoxia.

Box 3—Factors that suggest a cause of cerebral palsy other than acute intrapartum hypoxia

- Umbilical arterial base deficit less than 12 mmol/l or pH greater than 7.00
- Infants with major or multiple congenital or metabolic abnormalities
- Central nervous system or systemic infection
- Early imaging evidence of longstanding neurological abnormalities—for example, ventriculomegaly, porencephaly, multicystic encephalomalacia
- Infants with signs of intrauterine growth restriction
- Reduced fetal heart rate variability from the onset of labour
- Microcephaly at birth (head circumference less than a third of the centile)
- Major antenatal placental abruption
- Extensive chorioamnionitis
- Congenital coagulation disorders in the child
- Presence of other major antenatal risk factors for cerebral palsy—for example, preterm birth at less than 34 weeks' gestation, multiple pregnancy, or autoimmune disease
- Presence of major postnatal risk factors for cerebral palsy—for example, postnatal encephalitis, prolonged hypotension, or hypoxia due to severe respiratory disease
- A sibling with cerebral palsy, especially of the same type

Evidence for possible antenatal causes of cerebral palsy

Possible antenatal causes of neurological impairment are listed in box 3. The presence of any one of these factors greatly reduces the likelihood that acute intrapartum hypoxia was the cause, or the sole cause, of any subsequent neurological impairment. The absence of any of these factors does not exclude an antenatal cause, as much of the evidence can be very difficult to ascertain retrospectively.

Tests and signs of less predictive value

Meconium staining of amniotic fluid

It is not possible reliably, either by inspection or by placental histopathology, to distinguish clearly between old and fresh meconium staining of amniotic fluid, nor to date the time interval between meconium passage and birth. Oligohydramnios increases the concentration of any meconium in amniotic fluid. Oligohydramnios may be associated with intrauterine growth restriction.

Placental pathology and the timing of hypoxic-ischaemic cerebral injury

At present there are few methodologically rigorous studies to link various forms of chronic placental pathology and long term neurodevelopmental outcomes despite numerous studies of candidate lesions.[w64] Recent suggestions that thrombosis in large fetal vessels in the placenta can be linked to ischaemic cerebral lesions and cerebral palsy merit further investigation in appropriately designed studies of adequate size.[w65] [w66] However, it would be unjustifiable to conclude that the absence in any particular case of any of the chronic placental lesions, which by their nature could reasonably be assumed to have antedated labour, necessarily means that an hypoxic injury must have occurred during labour.

Are neurological sequelae preventable in cases of excessive intrapartum hypoxia?

It is not possible to ascertain retrospectively whether earlier obstetric intervention could have prevented cerebral damage in any individual case where no detectable sentinel hypoxic event occurred. After a detectable sentinel hypoxic event, it is necessary to consider the local conditions and facilities available at the time of the birth in question when commenting on

whether the care provided met acceptable standards. Any major deviations from the range of normal clinical responses can only be considered critical to the development of cerebral palsy if they could plausibly and most likely have affected the duration or severity of the hypoxic event. The actual length of time and degree of hypoxia required to produce cerebral palsy in a previously healthy human fetus is not known. Many special physiological mechanisms protect the fetus from acute hypoxia, allowing it to survive intact for a longer period—minutes to perhaps hours—than an adult with similar blood gas concentrations.[w48] The questions in box 4 should be posed when trying to identify possibly preventable causes of cerebral palsy during pregnancy, labour, and delivery. Standards of care should be dictated by systematic review of high quality research.[w67]

Who should be an expert witness in cases of cerebral palsy?

No one person is an expert in all the facets of cerebral palsy. This task force has therefore drawn on a wide variety of disciplines for contributions to this statement. Clearly, experts should keep to their own area of expertise[w68] and should not be tempted to offer opinions in areas in which they are not qualified.[w69] A peer in obstetrics is the best person to assess the clinical management of an obstetrician, and a neonatologist the neonatal management. A variety of evidence based opinions may be needed to elucidate further the possible causes of cerebral palsy in a particular case.[w70] The appropriate eligibility criteria to offer expert medicolegal clinical opinion regarding cerebral palsy are detailed in box 5.

Conclusion

This international consensus statement has been prepared to help the public, healthcare workers, those researching in this area, and, where necessary, courts of law to understand more easily the probability of whether, in any particular case, there is convincing evidence to suggest that the pathology causing cerebral palsy occurred during labour and whether it was reasonably preventable. Recent research strongly suggests that the large majority of neurological pathologies causing cerebral palsy occur as a result of multifactorial and mostly unpreventable reasons during either fetal development or the neonatal period.

Box 4—Questions pertinent to assessing the preventability of cerebral palsy assumed to be due to an acute intrapartum event

Were there risk factors for an antenatal cause of cerebral palsy?
Was there a sentinel hypoxic event?
Was there an intervention available proved to reduce the rate of cerebral palsy?[w68]
Have the criteria for defining an acute intrapartum hypoxic event been met?
Could the signs of fetal compromise reasonably have been detected?
Was there an avoidable major delay in expediting delivery?
Would quicker delivery of the baby have compromised the mother's health or life?
Would an earlier delivery, if practical, have prevented or ameliorated the outcome?

Box 5—Recommendations for expert witnesses giving evidence on cerebral palsy causation

Choice of witness
The witness must have full qualifications in the area in which he or she is giving an opinion
The witness must have credibility with respect to his or her knowledge among his or her peers proved, for example, by relevant research published in peer reviewed journals, by ongoing continuing education, and by quality assurance activities
In most circumstances the witness should still be practising in that area, should show evidence of being familiar with the current relevant literature, and should have a reasonable understanding of the local conditions and facilities at the time of the birth in question

Conduct of witness
The witness should avoid giving specific opinions outside of his or her expertise
Experts should advise: *(a)* of the spectrum of care considered reasonable by their profession at the time of the birth, and *(b)* of all the options in any clinical situation rather than only of the ideal option in the best of circumstances with the advantage of hindsight
An expert should not be an advocate for either party but should advise and educate on the scientific and clinical validity or otherwise of the evidence

We thank all those who have donated their time, expertise, and support to the compilation of this consensus statement. This debate was initiated by the Perinatal Society of Australia and New Zealand, and the consensus statement is supported by several colleges and scientific societies (box 1).

Endpiece

The whole idea was so disgusting

At first he was very discouraging, to my astonishment then, but now I fancy he did it as a forlorn hope to check me; he said the whole idea was so disgusting that he could not entertain it for a moment. I asked what there was to make doctoring more disgusting than nursing, which women were always doing, and which ladies had done publicly in the Crimea. He could not tell me. When I felt rather overcome with his opposition, I said as firmly as I could, that I must have this or something else, that I could not live without some real work, and then he objected that it would take seven years before I could practise. I said if it were seven years I should then be little more than 31 years old and able to work for twenty years probably. I think he will probably come round in time, I mean to renew the subject pretty often.

Elizabeth Garrett Anderson, in 1860, on her father's reaction to the news that she wanted to train as a doctor